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Experimental Oak Wilt Control In Missouri

T. W. JONES AND T. W. BRETZ

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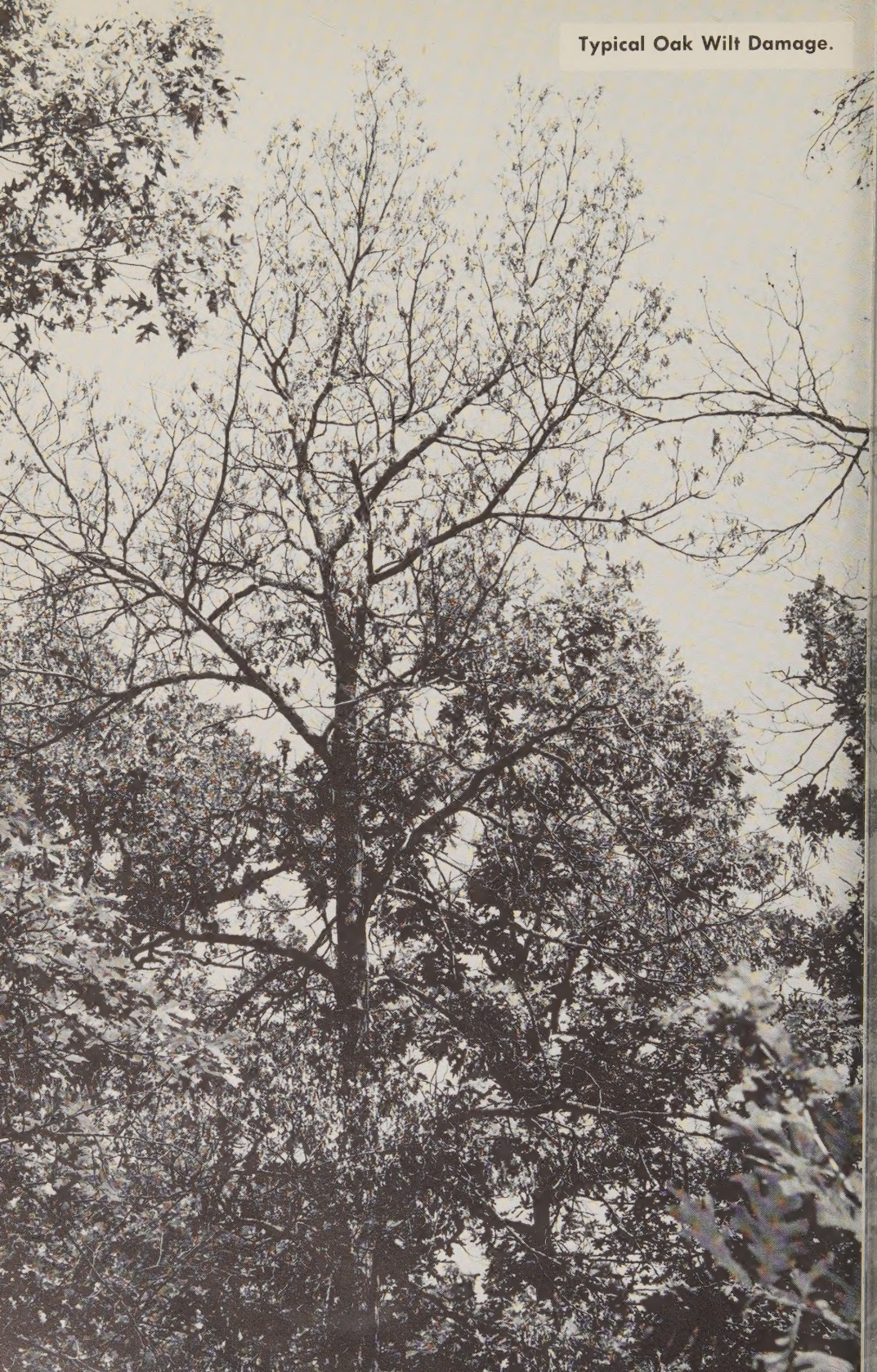

Typical Oak Wilt Damage.

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A report on School of Forestry research project 52, Oak Wilt

Experimental Oak Wilt Control In Missouri

by T. W. Jones and T. W. Bretz[1]

INTRODUCTION

Considerable attention is being given by a number of Federal and State research groups in the eastern United States to the development of effective measures for the control of oak wilt. It has been determined that the causal fungus, *Ceratocystis fagacearum* (Bretz) Hunt, may move from diseased trees to nearby healthy trees through natural root grafts and account for some local spread of the pathogen (*9*). The fungus also spreads over greater distances, but the method by which it spreads has not been definitely established. It is known that the fungus will develop spore mats under the bark of some wilt-killed trees. Such mats, exposed by cracking of the bark, are believed by some investigators to constitute an important source of inoculum for the above-ground, "long-distance" spread of the fungus (*3, 4, 5, 7, 11, 14, 15*). There is also evidence that bark and wood-boring beetles may be involved in "long-distance" spread of the fungus in the absence of mats (*1, 2, 13*).

Prevention of underground spread of the fungus through root grafts and prevention of its spread from the above ground parts of the tree have formed the bases of the oak wilt control measures presently recommended (*5, 6, 7, 14*). A cooperative[2] oak wilt control study, utilizing the principles of sanitation and/or isolation of diseased trees, was conducted in Missouri from 1952 through 1956 to test the efficacy of these measures under Ozark conditions. Results of those tests provide the bases for this report.

PROCEDURE

The study was conducted on 20, square, 40-acre plots on which various treatments were applied and on 20 plots, totaling 2135 acres, on which no control treatments were applied. The latter consisted of strips 10 chains wide around each of the 40-acre plots. These were not check plots, in a strict statistical sense, and were not treated as such in the analysis of results, but they did provide an extensive infected area on which uncontrolled disease development was measured. All the plots were located in second growth, oak-hickory stands in a 10-county area in southeastern Missouri. They included ridgetop, slope, and bottomland sites. Oak wilt occurs throughout this part of the State and from one to several actively wilting diseased trees were present on each plot when it was established.

[1]Respectively, Pathologist, Columbia Forest Research Center, Central States Forest Experiment Station, Forest Service, U. S. Department of Agriculture, Columbia, Missouri, and Professor of Forestry, School of Forestry, University of Missouri, Columbia, Missouri.

[2]Central States Forest Experiment Station, Forest Service, U. S. Department of Agriculture, Columbia, Missouri, and School of Forestry and Department of Botany, University of Missouri, Columbia, Missouri. This study has been financed in part by grants from the National Oak Wilt Research Committee, Memphis, Tennessee and the Division of Forestry, Missouri Conservation Commission, Jefferson City, Missouri.

A 100 percent cruise was made of each plot once each year during June or July. Survey crewmen, spaced at one-chain intervals, systematically searched each plot for diseased trees. Symptomatic trees were tagged and sampled for laboratory confirmation of the field diagnosis. Their location was mapped and species, d.b.h., height, crown class, and degree of defoliation recorded.

The following treatments, designed to prevent underground spread of the fungus through root grafts or to eliminate the source of inoculum for aboveground spread, were tested.

Isolation of Diseased Trees: On seven of the plots, all infection centers[3] found in 1952 and in each year thereafter, received the isolation treatment. In this treatment, all oaks within 50 feet of diseased trees were poisoned, regardless of species. The diseased oaks were left standing.

Sanitation of Diseased Trees: Seven other plots were untreated in 1952, but starting in 1953 and every year thereafter, all infection centers found on these plots received the sanitation treatment. In this treatment only the diseased trees were cut and burned.

Isolation-Sanitation of Diseased Trees: On six other plots, all infection centers found in 1952 and in each year thereafter, received the isolation and sanitation treatment. In this treatment all oaks within 50 feet of diseased trees were poisoned and the diseased trees were cut and burned.

All control measures were applied each year during August. The tree poisoning procedure employed in the isolation operation in 1952, consisted of applying a 4-pound-per-gallon solution of Ammate in water in basal frills in trees 1 inch d.b.h. and larger. A 4 percent solution of commercial 2,4,5-T in fuel oil was applied as a basal spray to trees with a d.b.h. of less than 1 inch. In 1953, and in each year thereafter, a 4 percent solution of commercial 2,4,5-T in fuel oil was used instead of Ammate in the treatment of trees 1 inch d.b.h. and larger. The treatment for the smaller trees remained unchanged.

Not all trees poisoned in the isolation operation died. Although the initial applications of herbicide accomplished good top kill, 34 percent of the poisoned trees developed basal sprouts. All poisoned trees with living tops and/or sprouts one year after the initial treatment were repoisoned. This killed all tops and reduced the number of trees with living sprouts to 10 percent of the trees originally treated.

Diseased trees that were felled and burned in the sanitation operation were cut into small sections, piled on the stumps, and burned until the small wood was consumed and the larger pieces deeply and thoroughly charred. The fire was then extinguished with water.

RESULTS

During the five years that the study was in progress, 1809 diseased trees were located in 784 infection centers. The effectiveness of the control efforts has

[3]For the purpose of this study, an "infection center" was considered to be one to several trees wilting or dead from the effects of the disease, each of which was within 50 feet of another diseased tree in the group. If a gap in excess of 50 feet occurred, the group was divided at that point into two separate infection centers. An active infection center was one in which one or more trees were currently wilting.

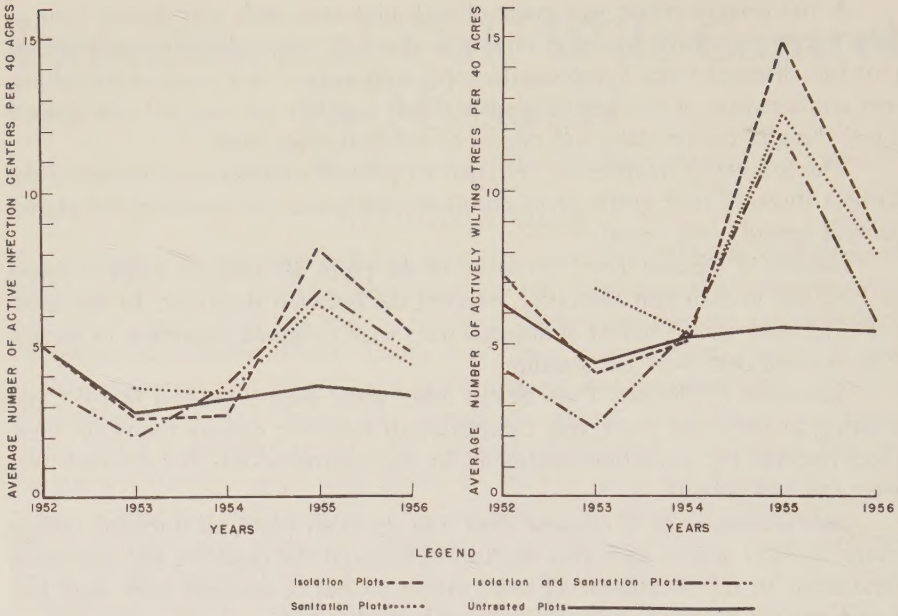

Fig. 1—Average number of active infection centers and wilting trees per 40 acres by treatments and years, 1952-56.

been evaluated on the basis of the number and location of infected trees and infection centers found each year on the treated and untreated plots and on the trend of these numbers.

The incidence of infection on the treated plots fluctuated considerably during the course of the study and was slightly greater in 1956 than it was when the treatments were started. No indication of consistent disease reduction is apparent. The disease situation on the untreated plots remained relatively constant and was slightly less severe in 1956 than in 1952 (Fig. 1).

A major portion of the wilting trees found each year was concentrated in the immediate vicinity of old centers. On all types of plots, 65 to 73 percent of the wilted trees were within 100 feet of a tree killed by the disease in a previous year. This concentration is particularly evident when it is recognized that the total area within 100 feet of diseased trees is much less than the balance of the area of the plots (Table 1).

TABLE 1--LOCATION OF OAK WILT TREES IN RELATION TO TREES INFECTED IN A PREVIOUS YEAR, 1953-56

Type of Treatment Applied on Plots	Distance from Diseased Tree to Nearest Tree Infected in a Previous Year		
	0-50 Feet	50-100 Feet	More than 100 Feet
	Percent	Percent	Percent
Sanitation	71	2	27
Isolation	19 1/	47	34
Isolation-sanitation	16 1/	55	29
Untreated	57	8	35

1/ Sprouts from stumps of herbicide treated trees.

Sanitation Treatment

Sanitation of diseased trees by burning the aboveground portions did not consistently reduce the amount of oak wilt on plots on which this treatment was applied. Whether the elimination of the aboveground source of inoculum reduced the amount of "long-distance" spread within the plots is questionable but if so, this was offset by the amount of local spread (up to 50 feet). An average of 71 percent of the infected trees found each year on these plots occurred within 50 feet of treated diseased trees.

Isolation and Isolation-Sanitation Treatments

The relative amount and location of oak wilt on the isolation and isolation-sanitation plots was very similar. Whether the diseased tree was cut and burned or left standing apparently had very little influence on results.

Both of these treatments reduced the apparent amount of local spread. Most of the tops and many of the root systems of the trees in the treated area were killed by the herbicide application but sprouts from the root systems which were not killed were present for symptomatic evidence of local spread. Through 1954, oak wilt symptoms were not observed on sprouts from trees poisoned in previous years, but in 1955, 31 infected sprouts were found in 17 of the 129 infection centers which had received one of these treatments up to that time. Ten of these infection centers had first been treated in 1952, three in 1953, and four in 1954. In 1956, thirty infected sprouts were found in 23 of the 226 infection centers which had received one of these treatments up to that time. Five of these infection centers had first been treated in 1952, six in 1953, six in 1954, and six in 1955.

The apparent reduction in local spread of disease on plots on which the isolation or isolation-sanitation treatments were applied was offset, in terms of overall oak wilt control, by the number of new infections in the 50-foot zone adjacent to poisoned areas (50 to 100 feet from treated diseased trees). Approximately 50 percent of the new infections each year since 1952 through 1956 occurred in this area. Approximately one-half of the infected trees in this zone occurred within 10 feet of the edge of the treated area, one-fourth were in the next 10 feet, and the remaining one-fourth were distributed in the remaining 30 feet. Thus it would seem that the greatest difference between the effect of isolation and isolation-sanitation treatments compared with sanitation alone and no treatment was that the former treatments caused the heaviest concentration of infection to occur 50 feet farther away from previously infected trees.

Root-Graft and Overland Disease Transmission

The relative frequency of oak wilt spread through root-grafts as opposed to overland transmission of the disease in Missouri is unknown. It has been assumed

that spread over distances in excess of 50 feet, with a few possible exceptions, should be attributed to aboveground transmission and that spread over distances up to 50 feet may be attributed to below-ground transmission, aboveground transmission, or both.

The pattern of oak wilt spread on the sanitation plots, where a high percentage of the diseased trees occurred within 50 feet of treated, diseased trees, could be explained by a preponderance of root-graft spread. Theoretically, the sanitation treatment would have little effect on this mode of fungus dissemination. On the other hand, if root-grafts are as prevalent in the area as this would suggest, one might expect more infections in a comparable zone on the untreated plots than was actually recorded. The concentration of infections could also be accounted for by short range, aboveground spread alone or in conjunction with root-graft transmission.

The pattern of oak wilt spread on the isolation and isolation-sanitation plots, where a high percentage of the diseased trees occurred more than 50 feet but not more than 100 feet from treated diseased trees, is less readily explained by root-graft transmission. The herbicide applications used to create the isolation zones around diseased trees resulted in top kill of nearly all treated trees but a portion of the root system of approximately one-third of these trees was not killed. It is possible that chains of root-graft connected trees, extensive enough to bridge 50 foot isolation zones, occur in forest stands. A competitive demand for water between such trees might also be assumed. Under such conditions, elimination of transpiration pull in the diseased tree and elimination of the transpiring crowns in the isolation zone might tend to accelerate the movement of water and fungus spores from the diseased tree through a chain of root-grafts into transpiring trees beyond the treated zone. On the other hand, untreated infection centers seldom enlarge to such an extent in Missouri, even over periods of several years.

In creating the isolation zone around diseased trees on these plots, an average of 244 healthy oaks, 1 inch d.b.h. and larger, were poisoned per infection center. This created openings in the forest canopy with minimum diameters of 100 feet in which there were many dead and dying trees. These areas proved highly attractive to large numbers of insects of various species, including the small oak bark beetle. Whether the unknown vector for the oak wilt fungus was among those insects is obviously not known, but if such should be the case, the high incidence of infection among the trees in the area immediately adjacent to the treated areas might be explained by short range vector transmission.

DISCUSSION

A number of factors which prevailed during the course of the study and which may have influenced the results of these tests should be considered.

The control measures were applied on 40-acre areas surrounded by infected

but untreated forest stands and the influence exerted by these outside infections on the treated plots is unknown. However, since wilting trees on the plots were concentrated each year in the immediate vicinity of old infection centers, it would seem that conditions exist at these old centers which predispose the surrounding trees to infection. The failure of the control efforts appears to be due more to failure to control the disease within the plots than to invasion by the disease from outside the plots. Conversely, these results suggest that measures which would *effectively* eliminate or neutralize local sources of inoculum, which would counteract the predisposing factors suggested above, could logically be applied to relatively limited areas even though surrounding infections are uncontrolled.

A single cruise was made of each plot each year between the middle of June and the end of July. A single annual survey will not detect all of the oak wilt infected trees in an area since some trees do not show disease symptoms until mid-summer or later and would be missed in surveys made prior to that time. However, the number of trees in this category are a very minor portion of the total number of symptomatic trees found in Missouri annually. The work within the plots connected with application of control measures, in effect, provided a partial resurvey of most of the plots in August of each year. Few additional diseased trees were located at this time.

Control treatments were applied each year during August and thus there were variable intervals of time between initial symptom expression, detection, and treatment of diseased trees. This delay may have reduced the effectiveness of the treatments since a general principle in plant disease control is that the sooner the control treatment is applied following infection the greater the chance for effective control. However, there are practical limitations to the frequency of examination of large areas of timberland and the single annual cruise and interval between infection and treatment were considered fairly realistic in terms of conditions which might well prevail in large scale control programs. Furthermore, the intensity and thoroughness of the one annual survey was considerably greater than could reasonably be attained in a large scale operation and this would tend to offset, at least in part, the possible influences of the factors discussed above.

The difficulties in field recognition of diseased trees of the white oak group complicates the detection phase of a control program. Seventeen percent of the diseased trees found in this study were species of this group. The criteria upon which tentative field identification of diseased white oaks was based allowed for much greater variability in symptom expression than those considered applicable to trees of the red oak group. Even so, it is probable that the number of infected white oaks which escaped detection considerably exceeded the number of non-detected, diseased red oaks. Some white oaks recover from the disease, at least for a period of several years and it has been reported that the fungus may remain in such trees in a viable condition for at least one year (*8, 11*). The only

foliar symptoms discernible in some white oaks from which the fungus was recovered were a few scattered brown leaves. A majority of the white oaks in the Ozark forests have a few brown leaves in the crown for one cause or another yet it has not been shown nor is it suspected that they are all wilt infected. The number of infected white oaks which display only obscure symptoms or none at all can only be speculated upon, but this virtually unrecognizable possible source of oak wilt inoculum must be considered as part of the oak wilt control problem.

The objective of the isolation treatment was to rapidly create a zone around the infected trees in which there were no living roots and thus root-graft spread of the fungus would be prevented. The herbicide applications used did not accomplish this objective. An average of 34 percent of the trees poisoned each year sprouted, indicating that at least a portion of the root system of such trees was alive. A second application of herbicide, one year after the initial application, reduced the number of trees with live sprouts to 10 percent of the number originally treated. 2,4,5-T in fuel oil was the herbicide selected for use after comprehensive study had shown it to be the best of various chemicals tested for killing all sizes and species of oaks in Missouri (10). The best results obtained in these tests was complete kill of an approximate 70 percent of the oaks treated. Greater tree killing effectiveness has been reported for C.M.U. and sodium arsenite, but due to the slow rate of kill of the former and the hazard to open range stock of the latter, these two materials were not used. Although the materials and methods used did not completely fulfill the objective of the treatment, it seems unlikely that this objective can be fully attained with the suitable herbicides presently available.

The objective of the sanitation treatment was to destroy the aboveground source of disease inoculum. The burning treatment applied unquestionably fulfilled this objective. Whether it was, or reasonably could be, applied soon enough following infection to be completely effective is subject to question. Also, no practicable means have been demonstrated for elimination of the fungus in the roots of diseased trees and the importance of this possible source of inoculum is unknown.

SUMMARY AND CONCLUSIONS

Several treatments, suggested as control measures for the oak wilt disease, were tested experimentally in Missouri for a period of five years. Isolation of diseased trees by poisoning surrounding healthy oaks, sanitation by cutting and burning diseased trees, and a combination of both treatments were applied on a number of 40-acre plots.

None of these measures, as applied in Missouri, resulted in control of the oak wilt disease. The number of trees infected with oak wilt each year on untreated plots remained relatively constant during the course of the study. The number of trees infected each year on the treated plots fluctuated erratically but no consistent improvement in the disease situation occurred.

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